Is oxygen limitation in warming waters a valid mechanism to explain decreased body sizes in aquatic ectotherms?

Running head: Oxygen limitation and "shrinking fish"

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Acknowledgements

The authors would like to acknowledge funding from Australian Research Council (grant No. DP170104240) and the Kone Foundation (to AA), Horizon 2020 European research projects ClimeFish (grant No. 677039) (to ARB) and Australian Academy of Science (to JRM)

Biosketch

The author team includes scientists working on physiology, ecological dynamics, and fisheries management, using both field-based and modelling approaches to develop a mechanistic understanding of climate change effects on individuals, populations and communities.

Global Ecology and Biogeography
A Journal of Macroecology

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Journal:	*Global Ecology and Biogeography*
Manuscript ID	GEB-2017-0247.R2
Manuscript Type:	Research Reviews
Keywords:	adaptation, alternative mechanisms, climate change, temperature size rule, growth, poikilotherm, energy budget, geometric biology

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Type of paper: **Concepts or Research Reviews**

Number of words in the abstract: **299**

Number of words in the main body: **8127**

Number of references: **90**

Abstract

Aim

The negative correlation between temperature and body size of ectothermic animals (broadly known as a temperature-size rule, or TSR) is a widely-observed biological pattern, especially in aquatic organisms. Studies have claimed that TSR arises due to a decrease in oxygen solubility and increasing metabolic costs at warmer temperatures, whereby an adequate oxygen supply to a large body mass becomes increasingly difficult. However, mixed empirical evidence has led to a controversy about the mechanisms affecting species' size and performance under varying temperature regimes.

Location

Global

Taxa

Aquatic ectotherms

Results

We briefly review the currently debated hypotheses and experimental data relating ectotherm body sizes to oxygen availability, and present them from an individual's energy budget perspective. We highlight the distinction between evolutionary and plastic mechanisms, and suggest that the oxygen limitation debate should aim to separate processes operating on short, decadal and millennial timescales. Further, we present the oxygen limitation debate in light of key alternative mechanisms proposed to explain the well-known TSR phenomenon.

Conclusions

A reconciliation among studies arguing for or against the importance of oxygen limitation might be achieved if the cost and trade-offs of oxygen supply are explicitly accounted for. Further, it seems that despite decades of research, we remain uncertain whether TSR is an adaptive response to temperature related to physiological (enzyme activity) and ecological (food, predation) changes, or to physiological constraints at the cellular level (oxygen supply and associated costs). Finally, we join in the call for collaborations between ecologists, physiologists, modellers and geneticists to develop a collaborative research program that would systematically assess the importance of competing hypotheses and their implications. To make reliable predictions about the future of marine ecosystems and devise best management responses, it is imperative that we understand the mechanisms driving body size responses to temperature changes in aquatic ectotherms.

Keywords: adaptation, alternative mechanisms, climate change, growth, poikilotherm, energy budget, geometric biology, temperature size rule

Introduction

Declining body size is recognised as a universal response of ectotherms to global warming (Daufresne et al. 2009). Body size reduction is particularly fast in aquatic environments (Forster et al. 2012, Horne et al. 2015), where sizes of fishes and other ectotherms have declined in the range of 5-20% over the last few decades (Baudron et al. 2014, Audzijonyte et al. 2016, van Rijn et al. 2017). Whilst harvest-induced changes in body sizes and growth rates (either phenotypic or evolutionary) are likely to be partly responsible (e.g. Sharpe & Hendry 2008, Audzijonyte et al. 2013), the rate of the observed decline seems much faster than expected from evolutionary responses alone (e.g. Audzijonyte et al. 2013) and in some species it does not correlate to the fishing mortality rate (Baudron et al. 2014). Instead, meta-analyses and other studies suggest that across a broad range of taxonomic groups (from bacteria to vertebrates) aquatic ectotherm body sizes decline by about 3% per 1°C of warming (Angilletta et al. 2004, Forster et al. 2012, Hoefnagel & Verberk 2015, Horn et al. 2015,). Yet, despite its ubiquity, we still do not adequately understand why animals should get smaller as temperatures rise and the quest for a general unifying mechanism remains one of biology's greatest challenges.

Oxygen limitation was originally proposed as one of the key mechanisms to explain smaller ectotherm body size at higher temperatures (see review in e.g. Atkinson et al. 2006). Since oxygen diffusion across membranes is less sensitive to temperature than metabolism ($Q_{10} \sim 1.4$ versus $Q_{10} \sim 1.5$-4.0 respectively, Woods 1999, where Q_{10} of 2 means that a process speeds up two-fold for every 10°C increase in temperature), smaller cells or body sizes help increase surface-to-volume ratio and improve diffusion-driven oxygen supply. Most multicellular organisms have more elaborate oxygen supply mechanisms than diffusion alone, yet the trade-offs in oxygen supply and demand and their relationship to body size have remained central to various hypotheses of temperature-dependent body size and performance optimisation (von Bertalanffy 1960, Pauly 1981, Portner et al. 2004, Atkinson et al. 2006, Verbek et al. 2011). For example, the gill oxygen limitation (GOL) hypothesis (Pauly 1981) proposes that body size in fish is limited by the inability of gills (whose surface area is limited) to supply sufficient oxygen to satisfy disproportionally increasing metabolic costs, which scale with body volume rather than surface area. Since metabolic costs increase at higher temperatures, it follows that the limitation on body size will be more pronounced in warmer waters. The temperature-dependent response of body tissues to oxygen supply is also central to a more general body size optimisation hypothesis, the MASROS ("maintain aerobic scope and regulate oxygen supply") (Atkinson et al. 2006). This states that through developmental plasticity, body size is optimised for a given environmental temperature to maintain the scope for aerobic activity. Oxygen is also

a key factor in the 'oxygen- and capacity-limited thermal tolerance' (OCLTT) hypothesis (Pörtner et al. 2017), which focuses on temperature-related aerobic scope and performance. While the OCLTT is only tangentially related to body size, it nonetheless presents oxygen supply as the main determinant of an organism's performance. The central tenet of all these hypotheses, that the ability to supply oxygen is limited and this limitation intensifies at larger body sizes and higher temperatures, is often invoked in ecological studies to explain observed decreases in body size (e.g. Baudron et al. 2014, Morrongiello et al. 2014, Waples & Audzijonyte 2016, van Rijn et al. 2017).

Recently, however, the importance of oxygen supply as a determinant of body size has been questioned. Lefevre et al. (2017, 2018) challenged the claim that oxygen supply could limit growth and body size under most conditions, at least for gill breathing ectotherms such as fish. Indeed, the current view among physiologists is that oxygen uptake can be easily modulated by organisms and therefore reflects oxygen demand rather than the other way around. The generality of OCLTT, and particularly the adequacy of aerobic scope curves to predict thermal performance, have also been debated (Jutfelt et al. 2018). In fact, the assumption of lower oxygen availability in warmer water itself is under scrutiny. Although oxygen solubility is lower at higher temperatures, the actual "bioavailability" is higher when the water viscosity, oxygen diffusivity and ventilation costs are taken into account (Verbek et al. 2011).

The confusion around the body size and temperature correlatons even extends to well-known "laws" and "rules" describing decreasing body sizes at warmer temperatures. For instance, the well-known Bergmann's rule was initially proposed to explain the interspecific pattern of larger endotherm body sizes in cooler environments, presumably driven by the physics of body surface to volume ratios and heat loss. Bergmann's rule focused on latitude, but was later applied to a range of geographic clines where temperature is only one source of variability. Originally the intraspecific extension of Bergmann's rule was referred to as James' rule (James 1970), but currently negative body size temperature correlations at both inter- and intra-specific levels, and for both endo- and ectotherms, are often referred to as Bergmann's rule (Meiri 2011). In parallel to these field observation-based rules, experimental studies have shown that temperature experienced during development also affects adult body sizes of ectotherms. In organisms as diverse as bacteria and fish, higher developmental temperatures lead to smaller adult body sizes, which was coined the name of temperature-size rule (TSR) (Atkinson, 1994). First, the TSR specifically addressed the phenotypic plasticity driven body size temperature correlation during the ontogenetic development. Subsequently, the TSR was applied to explain all temperature-size experimental findings (both phenotypic and genetic),

and sometimes even intra-specific field observations (e.g. Angilletta et al. 2004; Kozlowski et al. 2004).

Not surprisingly, recent debates about the possible role of oxygen limitation on species body size and performance, combined with the sheer vastness of literature on the topic, has left many ecologists and modellers confused about the validity of current approaches to predict species and ecosystem responses to climate change. Given that body size is a key determinant of intra- and interspecific interactions (Ohlberger & Fox 2013; Dell et al. 2011), demographic processes (e.g. Barneche et al. 2016) and fisheries productivity (Baudron et al. 2014), it is essential that the scientific community comes together to develop a coherent program to agree on and investigate alternative mechanisms behind body size responses to temperature. So far "progress toward a predictive theory [on species responses to environmental change] has been slowed by poor coordination between theoretical and empirical activities … Consequently, despite decades of intensive research, we have little hope of accurately predicting how populations, communities or ecosystems will respond to environmental change" (Angilletta & Sears 2011).

This review brings a new perspective on the possible roles of oxygen and temperature on the body size of aquatic ectotherm organisms by:

1) Suggesting that conflicting evidence about the role of oxygen on body size might be resolved if full costs and trade-offs associated with oxygen uptake are explicitly studied and taken into account;
2) Proposing a clearer distinction and recognition that body size reflects both genetic (evolutionary) as well as phenotypic (plastic) and epigenetic responses. The mechanisms involved in short-term acclimation are likely to differ from those that develop over longer evolutionary timescales. Broad scale inter-specific comparisons therefore may not be relevant for understanding species-specific responses to climate change over the next few decades (i.e. Lefevre et al. 2018 and Pauly & Cheung 2018 debate).
3) Highlighting a range of alternative mechanisms that could help resolve the apparently conflicting evidence for oxygen supply as a limiting factor on body size (Fig. 1). Body size is an emergent property of multiple intrinsic physiological (development rate, metabolic rate, intake rate, allocation to reproduction) and ecological (food availability, predation risk) processes, and oxygen supply is only one of them. Despite a large body of literature on the topic, these alternative mechanisms have not been clearly articulated and systematically tested.

A comprehensive review of all the alternative oxygen and temperature-driven mechanisms underpinning body size change is outside the scope of this paper, although we do hope for a collaborative effort to summarise current knowledge and identify knowledge gaps. To encourage such collaboration, we also propose key questions that should help to foster a deeper understanding of the underlying processes and more meaningful and accurate predictions.

Are aquatic organisms limited by their capacity to uptake oxygen, and what are the associated costs?

The fundamental question related to recent debate is whether, under normal environmental conditions (excluding extreme hypoxic environments) and normal activity levels, aquatic organisms at any size are limited by their ability to supply oxygen to body tissues. For example, the GOL hypothesis suggests that gill surface area has a smaller body mass scaling exponent than metabolism, because the effective surface area that can be supplied with adequate ventilation is limited by the physical space availability in an organism's gill region (Pauly et al. 1981, Pauly & Cheung 2018) (Fig. 1a). Even if gills were not limited by space to increase the surface area and ventilation rate, this activity itself requires oxygen and therefore cannot increase indefinitely (Pörtner 2002). According to Pauly & Cheung (2018), the GOL provides the most parsimonious explanation for the temperature-dependence of maximal attainable body masses in ectotherms, prevalence of small fish in tropical waters, higher sensitivity of larger individuals to temperature, and lower food assimilation efficiency in larger individuals (Pauly & Cheung, 2018).

From an evolutionary perspective, such inability to develop mechanisms for adequate oxygen supply seems rather unconvincing. Not only can gill surface area be rapidly modified, but other physiological mechanisms, such as cardiac output or blood oxygen affinity, should ensure that oxygen supply meets demand thereby avoiding non-adaptive growth responses (e.g. Lefevre et al. 2017, 2018). Nevertheless, a number of experimental studies and field observations do show a negative relationship between water oxygen concentration and ectotherm body sizes, both in fish and invertebrates. Guppies reared at 65% air saturation (i.e. 65% of normoxia) matured earlier and had stunted growth (Diaz Pauli et al. 2017), and growth rate was also negatively correlated with oxygen concentration in tilapia, when fish were reared at ca. 20%, 35% and 75% of air saturation conditions (such oxygen concentrations do occur in natural tilapia habitats) (Kolding et al. 2008). Similarly, the amphipod *Asellus aquaticus* raised at warmer temperatures grew to smaller adult sizes only when oxygen was limited (Hoefnagel and Verberk 2015), and rotifers in low-oxygen lakes reached smaller body sizes than those in similar

temperature but well-oxygenated waters (Czarnoleski et al. 2015). In contrast, other studies show that oviparous fish can increase their mass-specific oxygen consumption by nearly 30% compared to post-spawning fish (Karamushko et al. 2002), suggesting that changes in oxygen supply are regulated by the internal demands rather than supply. Experiments on "gill remodelling" in fish demonstrate that gill area is often smaller than geometric constraints would allow and, in a number of species (including in adult individuals, which, according to GOL should be gill-size limited) could be increased within days if needed (Nilsson et al. 2012). However, once the original environmental conditions return, the gill area was again decreased and lamellae "reabsorbed" (Nilsson et al. 2012).

Such dynamic modification of gill surface area raises a key question, not clearly articulated in the recent oxygen limitation debate, concerning the potential cost (energetic and survival) of maintaining high capacity for oxygen uptake rate. These potential costs include increased energetic cost of maintaining ion homeostasis and water transport, increased exposure to toxic substances in the water, and increased risk of disease and parasitism (Nilsson et al. 2012). For example, fish with a high infestation of the trematode *Dactylogyrus* (a gill fluke) did not increase their gill surface area even when exposed to lower oxygen conditions, possibly because more gill surface area would result in a higher parasite load (Nilsson et al. 2012). Furthermore, maximum gill area is not necessarily advantageous, because oxygen in excess can become a toxic substance and organisms must balance the need for adequate oxygen supply against costs of oxidative stress (Verberk et al. 2013). The key question which emerges then is not whether aquatic ectotherms, and especially fish, have mechanisms to increase their oxygen uptake (they clearly do), but what are the potential costs and drawbacks of these adaptations on an individual's energy budget, emergent growth and fitness? Are the costs, and form, of these mechanisms consistent across species and body sizes, and how should they be accounted for when trying to predict species responses to climate change?

We currently lack good data on the costs of modifying and maintaining larger gill surface area in warmer and lower oxygen environments. The energy expenditure of maintaining ion homeostasis through gills has been estimated to account for 4-10% of the total energy budget (Lefevre et al. 2017). This is not insignificant and compares to, for example, an estimated ontogenetic average of 10-14% total energy allocation to growth, in Pacific bluefin tuna or Atlantic salmon (Nisbet et al. 2012). Changes in the gill membrane permeability might help to increase functional gill area without increasing ion exchange rate and energetic expenditure (Nilsson et al. 2012), but the costs of maintaining gill ventilation, and minimising the accumulation of parasites and toxic substances, remain. In fact, the energetic cost of oxygen

supply and ventilation might be a key determinant of polar gigantism in many aquatic invertebrates, because in cold and viscous water the relative energy expenditure of ventilation is higher for small individuals and hence growing to big size becomes beneficial (Verbek et al. 2013). Note, that this polar gigantism hypothesis completely reverses the traditional reasoning of oxygen limitation – it is not that oxygen availability leads to smaller sizes in warmer waters, but rather the costs of ventilation lead to larger sizes in colder waters.

So how can we determine whether oxygen availability limits body sizes in ectotherms? One approach is to conduct broad scale meta-analyses that compare body size – temperature correlations in terrestrial and aquatic habitats. Since extracting oxygen from water is much harder than from the atmosphere, stronger negative temperature - body size correlations in aquatic organisms would suggest (indirectly) that oxygen may have a limiting effect on growth. Two recent meta-analyses showed that negative temperature – body size correlations are indeed stronger in aquatic compared to terrestrial ectotherms (Forster et al. 2012, Horne et al. 2015). For example, for every 1^{o}C increase in ambient temperature, body size decline was ~ 3% in marine and freshwater species, but an order of magnitude lower (0.35%) in terrestrial taxa. However, it is worth mentioning that meta-analyses may be subject to analytical biases, as for example, Klok and Harrison (2013) failed to find this effect using similar datasets (see possible explanations in Horne et al., 2015).

Another approach to explore the impacts of oxygen limitation on body size is through controlled experiments, some of which have been reviewed above. Yet, it seems that at least for fish the experimental support on whether oxygen availability is likely to limit growth remains inconclusive. First, for understandable logistic reasons, most experiments have been conducted on small-sized invertebrates, which have different oxygen uptake mechanisms compared to those of fish. Second, experimental oxygen treatments are often extreme compared to the changes expected due to global warming (e.g. 10% and 150% of saturation in an experiment with rotifers, Walczyńska et al. 2015). Third, to understand processes that affect wild organisms, experiments should include months or years of acclimation time, and ideally account for epigenetic developmental control by rearing several generations in new experimental conditions (see below). Fourth, when oxygen bioavailability is taken into account (Verbek et al. 2011), the difference between experimental temperature treatments for small organisms might be insignificant or even reversed. Fifth, while experiments may demonstrate that growth is reduced at low oxygen concentrations, many of them still do not elucidate the underlying mechanism of whether growth reductions are due to limited oxygen supply (compromised ability to maintain metabolism and build new tissues) or simply increased energetic cost

associated with increased intake (and thus less energy left for growth). Some of these issues are already being addressed in specifically designed experiments (including by the authors of this study) and many new studies are underway, all of which should bring important new insights in the near future.

The role of acclimation and adaptation to ensure optimal oxygen supply

The recent debate about the role of oxygen limitation on body sizes is largely focused on the adequacy of predicting "shrinking fish" in response to global warming (e.g. Cheung et al. 2013). Yet, the GOL hypothesis, while predicting climate change effects on fish body sizes over the next 50 years (e.g. Cheung et al. 2013; Pauly & Cheung 2018), applies the same principles to comparisons across distinct species. Proponents suggest that a gill's ability to supply oxygen sets a universal, temperature-dependent "insurmountable constraint" on fish body sizes, and explains why the tropics are mostly inhabited by small fish species. Such a universal constraint appears unlikely given the range of physiological mechanisms available to increase oxygen uptake, and the presence of large fish in the tropics (see further details in Lefevre et al. 2017 and Pauly & Cheung 2018). Instead, the central question for ecologists, physiologists and modellers aiming to understand the impacts of climate change is whether the small increases in water temperature experienced over several generations impact the individual body size of a given species, not whether large fish can inhabit tropical waters. In other words, are expectations derived from broad inter-species comparisons relevant to predict intraspecific responses? Are the constraints and costs of evolutionarily and plastic adaptations and rapid phenotypic or developmental changes, comparable to those from long-term evolutionary adaptations?

Species respond to temperature changes through phenotypic plasticity (acclimation), maternal effects, and evolutionary changes (including evolution of plasticity). All of these processes will be important in modulating climate change responses, and all of them might have some impact on the attainable oxygen supply and associated costs.

1) Acclimation

Empirical data show that most aquatic organisms exhibit substantial phenotypic plasticity to acclimate to temperature changes within days or a few weeks (Seebacher et al. 2014). "Gill remodelling", discussed in previous sections, is one such example of acclimation to rapidly increase oxygen uptake rate. Likewise, many organisms can reduce (or acclimate) their

standard metabolic rate within a few weeks following an acute temperature change. The Q10 values measured over acute exposures to temperature are clearly unsuitable to predict and model climate change responses. For example, acclimation from 1 to 8 weeks in sculpin *Myoxocephalus scorpius* when exposed to a rise in temperature from 10 to 16°C reduced Q10 of standard metabolic rate from 2.4 to 1.0, i.e. acclimation completely compensated for the effect of temperature (but the recovery of aerobic scope was only partial, Sandblom et al. 2014). Perhaps our expectation of high baseline metabolic rates and hence high oxygen demand with warming waters may naively rely on results from experimental studies with insufficient acclimation (Lefevre et al. 2017)?

While some degree of acclimation is likely, Q10 values from acute and acclimation experiments demonstrate that post-acclimation Q10 across a range of physiological rates (cardiac, metabolic or locomotion) is still close to 2, and is even higher for metabolic rate (Seebacher et al. 2014, Lefevre et al. 2016). This means that although many aquatic organisms do show capacity for acclimation, their physiological rates have nevertheless already increased by ca. 20% over the last 20 years (Seebacher et al. 2014). The extent to which ectotherms can keep acclimating to changes in temperature within reasonable biochemical constraints and fitness costs will have important implications for climate change predictions, but this matter is yet to be resolved. Also unresolved are the possible differences (and costs) of acclimation across ontogenetic stages, and across species from different latitudes and temperature regimes. Generally, post-acclimation Q10 values are higher for high latitude species (Seebacher et al. 2014) suggesting lower acclimation abilities, but it is unclear whether such a difference reflects their lower thermal plasticity, or simply the different thermal consequences of temperature changes in hot versus cold environments (Payne & Smith 2017).

In summary, it seems unlikely that acclimation of metabolic rates alone will compensate for increased oxygen demands in warming waters. Post-acclimation Q10 values are still close to 2, suggesting that a few degrees of warming is likely to lead to a substantial increase in metabolic rates. Yet, even small changes in Q10 values will have large effects on most models, and better characterisation of individual and population variability in temperature dependence of multiple physiological rates (e.g. metabolic, assimilation, feeding, and growth rates) is urgently needed. In the absence of complete acclimation of metabolic rate with warming waters, we now examine the potential roles of epigenetics and evolution.

2) Epigenetic effects

We are just beginning to appreciate temperature-induced epigenetic mechanisms (although its effects have been known for longer, e.g. Tanasichuk & Ware 1987), but to our knowledge they have not yet been applied in models to predict species' responses to climate change. Temperature can leave an imprint at particular ontogenetic stages and set developmental trajectories. For example, Scott and Johnson (2012) showed that extreme temperatures during embryonic development of zebrafish (*Danio rerio*) had permanent lifelong effects on their acclimation capacity to temperature. These effects included enhanced plasticity, suggesting that individuals exposed to extreme thermal conditions at an early stage can better acclimate to temperature changes later in life. Other examples have been documented. For instance, the rearing temperature of European pearlside (*Rutilus meidingeri*) embryos affected subsequent muscle growth (Steinbacher et al. 2011), while eggs of Atlantic salmon (*Salmo salar*) exposed to higher temperature produced individuals exhibiting better growth in warmer temperatures (Finstad & Jonsson 2012). Similar observations were made on rotifer (*Lecane inermis*) where adult size was impacted by the temperature experienced by the mothers and embryos , highlighting the importance of maternal effects and egg development stage (Walczyńska et al. 2015a).

It is clear that the thermal regime experienced during development and incubation can determine temperature sensitivity later in life across both vertebrate and invertebrate species (Jonsson & Jonsson, 2014), and individuals affected by higher temperatures are likely to produce more temperature-resilient offspring. It follows then that climate change impact predictions about increased metabolic costs and large decreases in body sizes based on acute temperature exposure experiments may be overstated. Does this mean that metabolic Q10 values and oxygen demands in response to warming, estimated from inter-generational experiments will be even lower than currently expected, and are there potentially different mechanisms at play? What are the trade-offs of these epigenetic effects on other traits of species reproduction and performance and how should we account for them? These questions are yet to be addressed.

3) Evolution

The importance of evolutionary adaptations in the oxygen limitation debate has two key aspects. First, long term evolutionary changes mean that physiological and anatomical constraints inferred from broad comparisons of phylogenetically distinct species are unlikely to apply to short term changes over the next few generations. Second, predictions for the next 50 or 100 years still need to consider evolution that can occur over the course of several

generations. There is no doubt that species are already adapting to changing environmental conditions, although we have limited understanding on how such adaptations might occur and what exactly will be selected (Merilä & Hendry, 2014 Seebacher et al. 2014). Current models attempting to incorporate evolutionary adaptations to environmental change mostly assume random fluctuations in trait values or directional change at some specified or phylogenetically derived rate (e.g. Catullo et al. 2015). Traits, however, can be strongly correlated and the evolution of one trait (e.g. metabolic rate or capacity for growth) is likely to involve trade-offs with other traits (e.g. maximum activity level). Incorporating these trade-offs is essential for accurate predictions and our mechanistic understanding on the effects of temperature on body size, yet we are not aware of models that have explicitly explored them in the projections of marine ecosystem futures.

Some insights into relevant trait trade-offs can be gained from countergradient variation studies in aquatic and terrestrial ectotherms and endotherms. Countergradient variation means that "genetic and environmental influences on phenotypes oppose one another, thereby diminishing the change in mean trait expression across the [environmental] gradient" (Conover et al. 2009). In other words, it shows that genetic adaptations to environmental gradients modify physiological processes to increase fitness at a given temperature. A review of genetic clines reported at least 60 cases of countergradient variation in fishes, amphibians and insects, mostly related to physiological traits (Conover et al. 2009). In contrast, only 11 cases of cogradient variation (genetic and environmental influences are aligned and accentuate the change in trait value across the environmental gradient) were identified, mostly in morphological characters (Conover et al. 2009). The strength of countergradient clines matched well with the steepness of environmental gradients, suggesting that such variation might be ubiquitous (Baumann & Conover 2011). Collectively, these studies show that standard temperature-corrected physiological rates can vary significantly among populations or even individuals within one population (Burton et al. 2011; Dmitriev 2011) and that adaptive evolution to new temperatures can occur within a few generations (Barrett et al. 2011).

The associated trade-offs of such evolution in growth rate (and ultimately body size) may partly involve oxygen supply. For example, cold-adapted populations of silversides (*Menidia menidia*) had an almost twofold faster somatic growth, enabling them to reach similar body sizes during a shorter growing season (Baumann & Conover 2011). Fast growth was achieved by higher boldness, longer food search rate and bigger meals, but led to lower aerobic scope for sudden activity, poorer burst swimming ability and hence higher vulnerability to predation (Arnott et al. 2006, Norin & Clark 2017). A similar negative correlation between growth rate and

swimming performance was shown in experimental manipulations of three-spined sticklebacks *Gasterosteus aculeatus* (Lee et al. 2010), and even without predation, fast growth rate is known to affect other traits such as immune function (Dmitriew 2011).

In summary, evolutionary adaptations may help overcome any physiological constraints and optimise body sizes, and this is likely to be already happening. However, countergradient studies discussed above generally focus on a population's ability to increase growth rate in cold water environments with short seasons. It is hard to know whether the same mechanisms apply for optimising growth rate at increasing temperatures. Moreover, despite the prevalence of countegradient examples, comparisons of experimentally observed TSR patterns often correspond with the empirically observed Bergmann's clines, and are strongest in aquatic environments (Horne et al. 2015). Does this mean that countegradient adaptation is not strong enough to balance out increasing metabolic or oxygen demands in warmer temperatures when the full range of costs is accounted for? Or is a smaller body size in warmer waters (or larger sizes in colder waters) indeed optimal for reasons unrelated to oxygen, where developmental TSR reflects long term evolution of plasticity to optimise performance in the expected environment?

Alternative explanations for the temperature-size rule and their relation with oxygen

While the debate on the role of oxygen availability as a limiting factor for ectotherm body sizes still appears inconclusive, it also fails to acknowledge a range of alternative and widely researched mechanisms proposed to explain the ubiquitous temperature-size rules (Bergmann's, James' or TSR in a more narrow sense). Adult body size is a trait that emerges from a range of interacting factors that directly and indirectly affect the growth trajectory. The mechanisms leading to negative body size-temperature correlations can be both intrinsic (i.e. genetic, physiological) and extrinsic (i.e. environmental, ecological) to the individual (Fig. 1b-f). The intrinsic processes may involve, for example, the temperature dependence of metabolism and hormonal effects (Reinecke et al. 2005), while the extrinsic processes may entail predatory avoidance, pollution and nutrition (Jobling & Baardvik 1994). These mechanisms can be determined by genetic architecture of life-history strategies, plastic growth responses, or the evolution of plasticity itself (Seebacher et al. 2014). It is conceivable that oxygen might play a direct or indirect role in some or even most intrinsic and extrinsic mechanisms, but convincing empirical evidence is often lacking. Below we highlight the main categories of mechanisms that have been proposed to explain a negative temperature – body size correlation. Rigorous and systematic evaluation of these mechanisms with empirical data is urgently needed to illuminate

long standing controversies, and bridge currently parallel and potentially isolated scientific hypotheses and disciplines, criticised by Angilletta and Sears (2011) or Lefevre et al. (2017).

1) Intrinsic physiological models suggested to explain the body size and temperature correlation

1. Decoupling of developmental and somatic growth rates

One of the main hypotheses used to explain the TSR is the mismatch, or different slopes, of temperature dependence in developmental rates (cell division or increase in life stage per time) versus growth rates (cell growth or increase in weight per time) (van der Have & de Jong 1996; Zuo et al. 2012; Forster and Hirst 2012). The biophysical model of van der Have & de Jong (1996) aims to provide a universal mechanism that could be applied across single to multicellular organisms by pointing to different molecular weights and/or different temperature sensitivity (activation energies) of molecules responsible for growth or protein synthesis (RNA subunits) and cell division (DNA polymerase). If growth and development are primarily determined by the activity of these molecules, then different temperature sensitivities will lead to changes in size (either positive or negative) with temperature (Fig. 1b).

While some cells do indeed become smaller at higher temperatures, this response is far from universal across different tissues or organs (Atkinson et al. 2006). Yet the mismatch between development and growth rates is indeed seen in many organisms, and nicely demonstrated in an experimental study of a brine shrimp *Artemia franciscana* (Forster & Hirst 2012). For this species, and other crustaceans, the slope of weight-specific growth rates against temperature decreases with the progression of life stages, meaning that higher temperature depresses growth in later life stages more than it does in early ones. In contrast, the slope of developmental rate against temperature is constant, and the rate of differentiation is not affected by an ontogeny-temperature interaction. Such responses produce a reverse TSR (larger body sizes at warmer temperatures) in the youngest life stages and regular TSR in adults. Further empirical support comes from many groups, including fish, and across several generations (Atkinson et al. 2006; Forster & Hirst 2012).

These empirical observations, although well supported, still do not inform us about the possible underlying physiological mechanism(s) of the temperature - body size relationship. The key assumption, that the main driver is different temperature sensitivities of developmental and growth enzymes or molecules (van der Have & de Jong 1996, Zuo et al. 2012), to the best of our

knowledge, remains empirically untested. Since developmental rates are tightly linked with size, decreased growth rates in later stages with temperature could also in theory be caused by compromised oxygen supply (in line with Pauly & Cheung 2018 arguments) or adaptive plasticity in expectation of such limitation.

2. Temperature dependence of growth efficiency

Growth efficiency is defined as the fraction of consumed (gross efficiency) or assimilated (net efficiency) energy incorporated as new body mass. First attempts to find mechanistic explanations for TSR were largely based on the argument that within species, the gross growth efficiency decreases with temperature (von Bertalanffy 1960, Strong & Daborn 1980, Perrin 1995) (Fig. 1c). These explanations largely rely on the von Bertalanffy growth equation, which models growth, as a function of anabolism and catabolism:

$$dw/dt = kW^m - lW^n$$

where W = body weight, k is the coefficient of anabolism, l is the coefficient of catabolism and m and n are exponent parameters. From this equation Perrin (1995) and Strong & Daborn (1980) suggested mutually exclusive mechanisms on how temperature, based on its effects on growth efficiency, could produce the TSR. The former one required different temperature dependencies of catabolism and metabolism constants (k and l), while the latter was based on changes in allometries of anabolism and catabolism (different m and n). Neither of these two theories seem to be sufficient. To explain the ontogeny-dependent TSR observed in crustaceans (Forster & Hirst 2012), both the constants and allometries have to change. Moreover, the meta-analysis of 97 laboratory experiments across a range of ectotherm taxa showed that growth efficiency in fact increased or was independent of temperature within biologically-relevant temperature ranges (Angilletta & Dunham 2003). Temperature-dependent growth efficiency therefore does not seem to explain the TSR.

In another recent meta-analysis across multiple species, Barneche and Allen (2018) reported indirect evidence that the fraction of resting metabolic energy that is allocated to growth (i.e. the "cost of growth") increases with temperature but is independent of size. This means that growth across all sizes and the trophic transfer efficiency in the ecosystem, becomes increasingly inefficient as temperature goes up. If the total available energy remains the same, increasing cost of growth will lead to less energy converted to biomass and smaller body size. Although at first the results of Angilletta and Dunham (2003) may seem contradictory to those

of Barneche and Allen (2018), we note that they are not necessarily comparable for two reasons. First, the results of Barneche and Allen (2018) are based on an inter-specific comparison with family-level parameter estimates which may or may not reflect the response that occurs within species. Second, it is possible that the increased costs of growth could come at the expense of other components of total metabolic rates (see energy budget figure 1 in Hou et al. 2008) without affecting the ratio between assimilated energy and growth.

One key problem with the growth efficiency approaches that rely on a von Bertalanffy function (Strong & Daborn 1980, Perrin 1995, Pauly & Cheung 2018) is that they ignore the single evolutionary goal of every organism – reproduction. The von Bertalanffy equation may describe asymptotic growth statistically, but it is not suitable for mechanistic understanding because it does not differentiate between growth and reproduction. Indeed, "the use of Bertalanffy's (1960) model of growth has been one of the main obstacles to a proper understanding of the factors responsible for the ubiquity of the temperature-size rule" (Kozłowski et al. 2004). To produce asymptotic growth the model requires that the exponent of catabolism is larger than the exponent of anabolism, but such a relationship is not universal across animals (e.g. Brown et al. 2004). Moreover, as already pointed out by Kozlowski et al. (2004), the attempt to understand asymptotic size based on anabolism and catabolism does not make evolutionary sense – why grow to a size where catabolism equals anabolism and no energetic surplus is left for reproduction? In many ectotherms, and especially in fish, reproductive output scales hyper-allometrically with size (Hixon et al. 2013, Barneche et al. 2018), an outcome that directly challenges the idea that growth is limited by increasing catabolic costs.

In summary, despite decades of research it is still unclear how the allocation of energy to different processes (metabolism, growth, reproduction), and their respective efficiencies, relates to size and temperature, and what the underlying mechanisms are. There is some support for different temperature-dependent allometric exponents of intake and metabolism across fishes (e.g. Lindmark et al. 2018) which could be due to surface-volume ratio effects or changes in water viscosity and respiratory costs. If, after accounting for reproductive allocation, energy conversion efficiency to growth is indeed lower at higher temperatures and larger sizes, does oxygen supply play a role? To answer this question we again need specifically designed experiments that control for temperature and oxygen and assess the full energy budget of individuals.

3. TSR due to larger reproductive output and cost

When energy expenditure for reproduction is considered, TSR could emerge if faster, earlier growth and /or developmental rate and earlier onset of maturation produces an overall larger lifelong allocation of energy to reproduction versus growth (Fig. 1d). This has already been proposed by Berrigan and Charnov (1994), who suggested that TSR results from a negative correlation between maximum body size and asymptotic growth rate, meaning that faster growth early in life leads to earlier maturation and smaller adult body size. Such a negative correlation is indeed observed across a range of taxa, and at least in fish has been formalised as one of the life-history invariants (Charnov et al. 2013). Since individuals start allocating to reproduction before the onset of maturation (gonadal development, reproductive behaviour and other associated costs), the slowing down of somatic growth rates should begin in the later stages of immaturity but not in juveniles, a pattern consistent with opposite temperature-size patterns at different ontogenetic stages (Forster & Hirst 2012). Moreover, reproduction entails not only the energy directly released in spawn, but also (possibly substantial) indirect energetic costs for energy conversion and reproductive behaviour (Audzijonyte & Richards 2018). These indirect costs will affect the final energy conversion rate, but cannot be directly estimated from the released egg weight and, typically, are not incorporated into growth models.

Higher overall reproductive allocation due to earlier maturation at higher temperatures can produce the TSR. However, the underlying mechanism, adaptive significance, and the role of oxygen for this phenomenon remain unclear. For example, if intake and metabolism allometries indeed reduce energy conversion efficiency at higher temperatures, smaller size and earlier reproduction will be an adaptive way to increase reproductive output. Alternatively, if oxygen supply to large body size is indeed compromised at higher temperatures, earlier maturation and resulting smaller body size would also be adaptive. This might suggest a potential role of oxygen concentration in the onset of maturation, which could be tested in experiments. As mentioned earlier, these questions should be addressed with experiments that assess detailed energy budgets (estimating growth and reproduction allocation and costs) under controlled temperature and oxygen conditions.

4. Changes in genome size

An alternative "bottom-up" explanation for the family of temperature-size rules is that colder temperature leads to increased genome size and consequently larger cells and slower cell division (Hessen et al. 2013). Changes in genome size could arise due to adaptation to cold conditions (e.g. genome duplication to increase enzyme activity levels) or maladaptive processes (accumulation of "junk DNA" in cold water due to smaller population sizes and

selection pressure). Experimental data and convincing proof for this hypothesis is thus far lacking, because, like with other hypotheses, such experiments would have to address possible genotype/environment interactions and their adaptive significance. However, it is important to note that, first, not all cells are larger in colder environments (e.g. Atkinson et al. 2006), and, second, that the "junk DNA" and selection-driven changes on the genome size would require several orders of magnitude of difference in population size (e.g. Lynch & Conery 2003). Since TSR is observed repeatedly within each generation (Forster & Hirst 2012), it should be relatively easy to assess how both cell and genome size change depending on rearing temperature.

2) Ecological processes that could lead to an emergent correlation between temperature and body size

Increased temperatures may cascade to alter resource levels, population dynamics and species interactions. For example, predator-induced changes in resource demand or supply could act to both increase or decrease the body mass of prey (DeLong & Walsh 2015). Experimental studies usually do not address these ecological factors, nor the likelihood that predator avoidance may substantially modify individual physiology. A broad range of ecological processes and their interactions with genotype and emergent growth makes predictions challenging. Nevertheless, two ecological processes seem to be sufficiently general to be considered as alternative candidates for the mechanisms underlying the temperature-size rule.

1. Mismatch in supply and demand of food availability

Resource supply models state that the proximate cause for optimal body size is determined by the temperature-dependent interplay of resource supply versus demand (Fig. 1e). This means that "optimal body size is that which matches bodily resource demand to the expected environmental supply of resources on a *per capita* basis" (DeLong 2012). If temperature affects the per capita resource demand and supply at different rates, then the optimal body size will also change. This could happen if, for example, metabolic rates (and subsequently food intake rates) increased faster than primary production rates, leading to a stronger control of consumers on primary producers (e.g. Schaum et al. 2018). Alternatively, changes in the ratio of protein and carbohydrate availability can be affected by different temperatures and subsequently affect adult body size, at least in terrestrial ectotherms (Lee et al. 2015). Moreover, even if resource density is temperature-independent, increased predation risk at high temperatures (see next section) may cause behavioural shifts in the prey that will inhibit

foraging (Lima & Dill 1990) and thus effectively reduce food supply and change body size (DeLong & Walsh 2015). This mechanism of food supply and demand is linked to external ecological conditions and is therefore different to the largely intrinsic oxygen supply/demand hypotheses discussed above. In general, the mechanism has good theoretical foundations, but so far the experimental evidence has mostly been derived from single cell organisms and remains inconclusive. For example, while experiments with a ciliate *Tetrahymena thermophilawhen* showed that food supply is linked to temperature, the body size response may take a wide range of forms (DeLong et al. 2017), which does not provide a universal explanation for the temperature-size rule.

2. Evolution of earlier maturation in response to increased mortality at higher temperatures

Across a range of environments, natural mortality generally increases with temperature (Pauly 1980). This selects for evolutionary changes towards earlier maturation and selection towards increased reproductive investment, which will in turn lead to smaller body sizes in warmer environments (Roff 2002, Kozlowski et al. 2004) (Fig. 1f). Note that this mechanism involves natural selection and evolution and is therefore different from the mechanism described in Fig. 1d, where earlier maturation is caused by developmental factors. Increases in natural mortality at higher temperatures could be driven by the direct effects of temperature (such as oxidative stress and faster senescence) or changes in feeding rates and predation mortality (Pauly 1980). While the former appears too small to explain TSR (Angilletta et al. 2004), latitudinal- or temperature-dependent changes in predation mortality can have a substantial effect on physiological adaptations, growth rate and body sizes (Lankford et al. 2001, Reznick et al. 1997).

Although this mechanism has strong support in life-history theory, evolutionary responses cannot explain developmentally driven TSR patterns within a single generation. Moreover, while evolutionary change of life-history traits can be rapid under strong experimental selection pressure (e.g. Conover & Munch 2002), the observed changes in ectotherm body sizes (10-20% change, e.g. Audzijonyte et al. 2013) and growth rates (e.g. 2.5% per annum, Morrongiello & Thresher, 2015) over the last few decades seem too fast to be explained by evolutionary change alone.

There is no doubt that natural mortality plays an important role in genetic and developmental growth trajectories (Lind & Cresswell 2005). However, the complex interplay of temperature, predation, resource availability and anti-predatory behaviour means that general predictions

are unlikely. We are still far from understanding the potential fitness consequences of anti-predatory behaviour (Lind & Cresswell 2005) and their links with an individual's physiology and even oxygen supply. For example, countergradient variation studies have shown that animals in colder and lower predation environments increase their meal sizes and thus maximise their growth rates, but have lower post-feeding aerobic scope for activity and therefore are more vulnerable to predation (Arnott et al. 2006). Yet, increase in feeding rates and natural mortality in warmer waters is more related to the overall productivity and activity rates, so it is unlikely that oxygen supply could be seen as a key underlying driver in determining optimal body sizes at different mortality regimes.

Conclusions and key future questions

It seems that despite each of the intrinsic and extrinsic mechanisms described above having some empirical support, 15 years after the Angilletta and Dunham (2003) review we are still reaching the same conclusion that none of these mechanisms appear to be sufficiently universal. Could the costs of oxygen supply in aquatic environments be an underlying driver for changes in growth efficiency, shifts in reproductive allocation, changes in cell and genome size, or ability to match intake rates with metabolism? Speculative links can be drawn, but we still do not have sufficient experimental data to confirm or refute the role of oxygen, either directly or through genotype/environment interactions, and evolution of developmental plasticity in driving observed patterns in body size. We also note that, for some species, experimental TSR studies show an increase rather than a decrease in size with increasing temperature (Atkinson et al. 1994, van der Have & de Jong 1996, Zuo et al. 2012). Yet, these exceptions are found mostly in terrestrial air-breathing organisms, suggesting that oxygen or any factor that differs between aquatic and terrestrial environments (e.g. viscosity) may play a role (e.g. Hoefnagel & Verberk 2015).

A resolution on the key processes that might shape individual body size with rising temperatures, and an understanding of the situations in which each will be important, requires interdisciplinary collaborations. We suggest that the outstanding research areas that must be addressed include:

1. Determining the importance of acclimation and epigenetic control of temperature dependence of metabolic rates and associated oxygen demand. How do temperature reaction norms change with acclimation at intra- and intergenerational levels? Is the change in temperature dependence different among different processes (e.g. search rate,

metabolic rate, escape rate, specific dynamic action and others; Dell et al. 2011, 2014), and how are they affected by body size (Lindmark et al. 2018)? Answers to these questions are urgently needed for all models that apply individual-level temperature dependence of physiological rates to predict population and community structure (e.g. Brown et al. 2004; Barneche et al. 2014).

2. Understanding the costs and benefits of increasing oxygen supply to meet higher metabolic demands in warmer waters. Are ventilation costs significant enough to affect energy availability for growth? And will increased oxygen uptake affect other functions, such as vulnerability to disease or predation? To answer these questions, we need more inter-generational experimental studies on both vertebrate and invertebrate ectotherms, in controlled oxygen and temperature conditions and with well quantified individual energy budgets. These studies would need to deal with realistic temperature and oxygen levels expected in the next century to be applicable for climate change predictions.
3. What are the adaptive or maladaptive implications of temperature-body mass correlations, and to what degree does selection work to account for potential constraints (enzyme rates, oxygen solubility) at molecular or cellular levels? Focused and well designed interdisciplinary studies are needed to answer these questions.
4. Do ectotherms living in regions experiencing different degrees of warming display decadal-scale changes in growth, body size and maturation consistent with projections from mechanisms outlined above? If the physiology underpinning the TSR is general, in the sense of affecting many species similarly, then there should be a sub-component of the total variation in growth (e.g. the common trend detected in Baudron et al. 2014) that is synchronous across species and also correlated with time trends in temperature. The long term data available from otoliths collected for commercial fish species represent a unique opportunity to use regional seas as laboratories for detecting the fingerprint of climate change (e.g. Morrongiello et al. 2012).

Figure legend

Figure 1. A simplified representation of possible and not exclusive mechanisms explaining the empirically observed phenomenon (top right) of decreasing ectotherm body sizes with increasing temperature. Blue symbols and lines indicate processes at lower temperature, while red indicates the same processes at higher temperature. Intrinsic mechanisms include: a) Oxygen limitation hypothesis (GOL, MASROS), where blue and red lines show rates of catabolism at cooler and warmer temperatures, and W∞ (red colour – warmer temperature)

shows the asymptotic weight determined by the difference between rates of oxygen supply and catabolism; b) different temperature dependence of DNA replication (development) and growth rates results in smaller cells and faster cell division at warmer temperatures; c) decreasing growth efficiency at higher temperature means that less energy is converted to growth (net growth energy - NGE) in relatively warmer environments; d) higher size-specific allocation to reproduction at higher temperatures (due to e.g. earlier maturation) leaves less energy for growth (growth energy - GE) in warmer environments; e) faster increase in energy demand (metabolism, activity cost, etc.) compared with food availability leaves different amounts of net energy (NE) for growth and reproduction in cooler and warmer environments; and f) increased predation mortality at higher temperatures drives an evolutionary response of higher net energy allocation to reproduction versus growth to ensure breeding occurs before an individual dies. Note that some panels have different units of x and y axes.

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